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Severe Traumatic Brain Injury: A Case Report

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Patient: **Male, 28**
Final Diagnosis: **Closed head injury**
Symptoms: **Bilateral mydriasis • coma**
Medication: **—**
Clinical Procedure: **Ventriculostomy and hemicraniectomy**
Specialty: **Neurology**

Objective: **Unusual clinical course**

Background: Traumatic brain injury remains a challenging and complicated disease process to care for, despite the advance of technology used to monitor and guide treatment. Currently, the mainstay of treatment is aimed at limiting secondary brain injury, with the help of multiple specialties in a critical care setting. Prognosis after TBI is often even more challenging than the treatment itself, although there are various exam and imaging findings that are associated with poor outcome. These findings are important because they can be used to guide families and loved ones when making decisions about goals of care.

Case Report: In this case report, we demonstrate the unanticipated recovery of a 28-year-old male patient who presented with a severe traumatic brain injury after being in a motorcycle accident without wearing a helmet. He presented with several exam and imaging findings that are statistically associated with increased mortality and morbidity.

Conclusions: The care of severe traumatic brain injuries is challenging and dynamic. This case highlights the unexpected recovery of a patient and serves as a reminder that there is variability among patients.

MeSH Keywords: **Brain Injuries • Clostridium difficile • Glasgow Coma Scale • Intracranial Pressure • Saline Solution, Hypertonic**

Abbreviations: **TBI** – traumatic brain injury; **ADLs** – activities of daily living; **GCS** – Glasgow Coma Scale; **GOS** – Glasgow Outcome Scale; **CT** – computed tomography; **ICP** – intracranial pressure; **CSF** – cerebral spinal fluid; **EVD** – external ventricular drain; **CPP** – cerebral perfusion pressure; **HD** – hospital day; **FI_{O2}** – fraction of inspired oxygen; **PEEP** – positive end expiratory pressure; **ARPV** – airway pressure release ventilation; **CMV** – continuous mandatory ventilation; **CPAP** – continuous positive airway pressure; **IV** – intravenous

Full-text PDF: <http://www.amjcaserep.com/abstract/index/idArt/897116>



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Background

In the United State alone, there are approximately 1.5 million traumatic brain injuries (TBI) per year, and TBI is the leading cause of death among individuals under the age of 45 [1,2]. Annually, these injuries result in approximately 50 000 deaths and about 80 000–90 000 cases of debilitating head injuries [2]. In the US, the estimated annual economic cost of TBIs is \$76.5 billion, and we must not forget the emotional and physical toll that disability inflicts on patients and their families [3]. In many cases, patients are left without the ability to work or to perform activities of daily living (ADLs) [4]. Initial management of TBI is the most critical time period because it will have the greatest effect on mortality and degree of debility that surviving patients will experience.

In TBI, the most important tool used to assess degree of brain injury and prognosis is exam findings. According to the guideline *Early Indicators of Prognosis in Severe Traumatic Brain Injury*, on average, 88% of patients who presented with bilaterally unreactive pupils became vegetative or died, and 4% had good recovery or moderate disability [5]. The Glasgow Coma Scale (GCS) uses exam findings to quantify level of consciousness following TBI, with 3 being the worst, defined as deep coma or death, and 15 being the best, a fully awake person. In a study by Fearnside et al., out of 315 patients with severe TBI, 65% with initial GCS of 3 died [6]. In a larger study by Marshall et al., out of 746 patients, 78.4% with initial GCS 3 died and 7.2% had mild to moderate disability [7].

Radiographic findings can also be used to predict morbidity and mortality, and can be used to guide surgical intervention. A review of 753 computed tomography (CT) studies that revealed abnormal mesencephalic cisterns, midline shift, and subarachnoid hemorrhage were associated with an increased risk of elevated intracranial pressure (ICP) and death [8]. Here, we describe a patient who had all of the above CT findings, and who presented with a GCS of 3 and bilaterally dilated and fixed pupils.

Case Report

Bystanders found a 28-year-old, unhelmeted, white male prone and unconscious after he had lost control of his motorcycle and went off the road. He was brought to the Emergency Department via ambulance intubated as a Level 1 Trauma activation. The physical examination revealed a GCS of 3T, 4 mm bilaterally fixed pupils, negative corneal response, right parietal cephalohematoma, and cerebral spinal fluid (CSF) otorrhea on the right. CT of the head showed subarachnoid hemorrhage with left frontal and temporal subdural hemorrhage (Figure 1), effacement of the suprasellar cistern (Figure 2), and effacement



Figure 1. 10/30/14 Presenting Head CT showing traumatic subarachnoid hemorrhage in sylvian fissure and left frontal and temporal subdural hemorrhage.

of the 3rd and 4th ventricles (Figure 3). In addition, CT studies showed a left frontal/temporal and parietal hematoma with mass effect and cerebral edema causing a 5.38-mm left to right midline shift (Figure 4), a frontal skull base fracture, and a complex non-displaced comminuted fracture of the right temporal bone. He was bradycardic, with his lowest heart rate recorded at 28 bpm, and hypertensive with an initial blood pressure of 172/118 mmHg and markedly elevated blood pressure of 221/105 mm Hg 30 min after his arrival at our facility. He required atropine push and nicardipine infusion. An arterial line and central venous catheters were placed for fluid and medication administration. Emergent treatment for herniation syndrome included endotracheal intubation, 30 grams of IV Mannitol, hypertonic solution of 23% (weight/volume) sodium chloride (NaCl), and left-sided decompressive craniectomy. Postoperatively, an external ventricular drain (EVD) was placed; the initial intracranial pressure (ICP) was 14 mmHg. The patient was examined postoperatively and also after EVD placement. His GCS was 5T, with bilaterally reactive pupils, and positive corneal reflex in the left eye. CT of his head showed improvement of midline shift (Figure 5) and the ventriculostomy catheter tip was found to be in the proper location in the frontal horn of the right lateral ventricle (Figure 6). The patient was then started on 3% NaCl continuous infusion. ICP and cerebral perfusion pressure (CPP) displayed normal values



Figure 2. 10/30/14 Presenting Head CT showing effaced suprasellar cistern and left temporal subdural hemorrhage.



Figure 3. 10/30/14 Presenting Head CT showing cerebral edema with effacement of third ventricle, bilateral traumatic subarachnoid hemorrhage, and left frontal/temporal subdural hematoma.

at 3–4 and 70–75 mm Hg, respectively, for the first 24 h with the EVD open at 10 cm H₂O.

On hospital day (HD) 2 the patient's ICP was elevated in the upper 20s in the setting of shivering, hypertension, and fever, which were controlled with increased sedation, nicardipine infusion, and targeted temperature management, respectively. Repeat CT revealed cerebral edema in evolution, with no worsening midline shift or hemorrhage.

On HD 3 his GCS improved from 6T to 8T, with intact brain stem reflexes. The next day, HD 4, the patient's oxygen saturation dropped from 96% to 87% requiring an increase in fraction of inspired oxygen (FIO₂) from 50% to 80% and positive end-expiratory pressure (PEEP). Respiratory cultures were obtained and a chest x-ray revealed worsening bibasilar opacities (Figure 7); therefore, he was empirically started on IV vancomycin.

On HD 5, the patient's GCS deteriorated from 8T to 3T in the setting of elevated ICP. During bronchoscopy, his ICP was noted to be markedly elevated at 46; therefore, 23% NaCl was administered intravenously over a period of 10 min. Subsequently, a repeat CT of the head was obtained that showed cerebral

edema in evolution and no change in midline shift or hemorrhage. Respiratory cultures on HD 6 revealed methicillin-sensitive *staphylococcus aureus* (>10 000 cfu/ml) and *pseudomonas aeruginosa* (>10 000cfu/ml). Repeat chest x-ray showed worsening infiltrates and bilateral pleural effusions (Figure 8). The antibiotic was switched from vancomycin to IV levofloxacin and IV cefepime.

Examination on HD 8 revealed an improved GCS of 6T. His ICP was controlled during an EVD clamp trial, and the EVD was then removed. There were also copious amounts of thick, tan endotracheal secretions; therefore, another bronchoscopy was performed using saline lavage. The patient was started on IV Zosyn and cefepime was discontinued. ICP was controlled by administering 23% NaCl. During a sedation vacation, his oxygen saturation decreased from 95% to 70% and the ventilator settings were therefore changed to airway pressure release ventilation (APRV) mode.

On HD 14, his oxygenation was improving and remained stable at 97%. The ventilator mode was weaned from APRV to continuous mandatory ventilation (CMV). The patient was taken off sedation. His GCS was 10T and he was started on amantadine.



Figure 4. 10/30/14 Presenting Head CT showing left frontal and parietal subdural hematoma, bilateral traumatic subarachnoid hemorrhage, and cerebral edema worse in left hemisphere with 5.38 mm of left to right midline shift.

He was started on oral metronidazole due to multiple loose stools positive for *Clostridium difficile* toxin B.

On HD 15, he began blinking to threat, with a GCS of 10T. A CT of his head showed improvement in diffuse cerebral edema and effacement of basal cisterns (Figure 9). He underwent a percutaneous tracheostomy with video-assisted bronchoscopy, and open gastrostomy tube placement by trauma surgery. The patient remained neurologically unchanged on HD 16, but repeat chest x-ray revealed an interval increase in right-sided infiltrates with resolution of left lung opacity. On HD 17, he continued to maintain adequate oxygen saturation at 97% and was started on continuous positive airway pressure (CPAP).

On HD 18 the patient developed tachypnea and he was placed back on CMV. The antibiotics were changed from Zosyn to levofloxacin and nebulized tobramycin due to piperacillin/tazobactam-resistant *Pseudomonas pneumonia*. On HD 19 his neurologic status was unchanged and he was started on Provigil. He was also given a protective helmet. Two days later, on HD 21, the patient's neurologic status remained unchanged and he was discharged to a long-term care facility.



Figure 5. 10/30/14 CT head after left craniectomy for decompression showing improved midline shift and evolving bilateral traumatic subarachnoid hemorrhage.



Figure 6. 10/30/14 Head CT after ventriculostomy showing catheter tip in frontal horn of right lateral ventricle.



Figure 7. 11/1/14 Chest x-ray showing bilateral interstitial infiltrate and left base atelectasis and pleural effusion.



Figure 8. 11/4/14 Chest x-ray showing worsening interstitial and alveolar infiltrates and pleural effusions bilaterally.

A follow-up visit three months later revealed the patient was living at home with his mother. In the interim his tracheostomy and gastrostomy tube had been removed. His major neurologic sequelae were transcortical motor aphasia and mood disorder. His GCS was 13 (E4, V3, M6). His GOS, on a 5-point scale, was 3, with severe injury and permanent need for help with daily living. His Modified Rankin Scale was 3 with moderate disability, requiring some help, but able to walk without assistance. His Lawton Instrumental Activities of Daily Living Scale was 4/8. Barthel Index was 95/100, and National Institutes of Health Stroke Scale was 5. Eleven months after the accident, he had similar outcome scores and had developed a seizure disorder; however, his speech was markedly



Figure 9. 11/14/14 New left occipital horn intraventricular hemorrhage, increased external herniation of left frontal contusion, and resolving diffuse cerebral edema with decreased effacement of third ventricle.



Figure 10. 8/3/15 Improved external herniation and increase in hydrocephalus secondary to prior brain injury.

improved with speech therapy. A repeat CT of the head showed improved external herniation (Figure 10).

Discussion

When predicting mortality and unfavorable outcome following TBI, exam, laboratory, and imaging findings can be used together by utilizing the CRASH and IMPACT calculators [9,10]. Unfavorable outcome is described as death, vegetative state, or severe disability. Here, we describe a patient who presented with a GCS of 3, bilaterally fixed pupils, and CT findings of subarachnoid bleeding, midline shift, subdural hematoma, effaced 3rd ventricle, effaced 4th ventricle, and effaced basal cisterns. Therefore, according to the CRASH calculator, which takes into account country, age, GCS, pupil reactivity, and CT findings, he had a 14-day mortality risk of 91.8% and a 95.7% chance of unfavorable outcome at 6 months [9]. His pertinent laboratory studies, which are utilized along with exam and imaging findings in the IMPACT calculator, revealed an initial glucose concentration of 260 mg/dL and a hemoglobin concentration of 15.4 g/dL. Using the IMPACT calculator, at 6 months, the patient's predicted probability of mortality was 62% and the probably of unfavorable outcome was 77%. He left our facility bedbound, ventilator- and tube feed-dependent, and in a minimally conscious state with a GCS 10T. Yet despite all this, he had a favorable recovery. Within 1 year of discharge he was able to live at home, interact, and go shopping with his mother, walk, feed himself, and perform simple chores and ADLs.

References:

1. Bruns J Jr, Hauser WA: The epidemiology of traumatic brain injury: A review. *Epilepsia*, 2003; 44: 2–10
2. Thurman DJ, Alverson C, Dunn KA et al: Traumatic brain injury in the United States: A public health perspective. *J Head Trauma Rehabil*, 1999; 14: 602–15
3. Shouten JW, Maas A. Epidemiology of trauma brain injury. *Youmans Neurological Surgery*; 2011; 6: 323
4. Humphreys I, Wood RL, Phillips CJ, Macey S: The costs of traumatic brain injury: a literature review. *Clinicoecon Outcomes Res*, 2013; 5: 281–87
5. Chesnut RM, Ghajar J, Maas AIR et al: Early indicators of prognosis in severe traumatic brain injury. *J Neurotrauma*, 2000; 17: 614–19
6. Fearnside MR, Cook RJ, McDougall P et al: The Westmead Head Injury Project outcome in severe head injury. A comparative analysis of pre-hospital, clinical, and CT variables. *BR J Neurosurg*, 1993; 7: 267–79
7. Marshall LF, Gattille T, Klauber MR et al: The outcome of severe closed head injury. *J Neurosurg (Suppl)*, 1991; 75: 28–36
8. Eisenberg HM, Howard GE, Aldrich EF et al: Initial CT findings in 753 patients with severe head injury. *J Neurosurg*, 1990; 73: 688–98
9. MRC CRASH Trial Collaborators, Perel P, Arango M, Clayton T et al: Predicting outcome after traumatic brain injury: practical prognostic models based on large cohort of international patients. *BMJ*, 2008; 7641: 425–29
10. Steyerberg EW, Mushkudiani N, Perel P et al: Predicting outcome after traumatic brain injury: Development and international validation of prognostic scores based on admission characteristics. *PLoS Med*, 2008; 5(8): 1251–61

This is a poignant reminder that variability between individual patients makes prognosticating after traumatic brain injury difficult and uncertain.

Conclusions

Our case shows that severe caution should be taken when using prior studies to make medical decisions about individual patients. Treatment of traumatic brain injuries is complex, and should continue to evolve with evidence-based medicine. Improvement in outcome is not based on 1 intervention; rather, it is the additive effect of multiple interventions. It is possible that the addition of multimodality monitoring could have further changed his outcome, and more studies need to be done to answer this. In addition, daily multidisciplinary rounds with Neurocritical Care, Trauma Critical Care, Infectious Disease, Pharmacy, Respiratory Therapy, Physical Therapy, Occupational Therapy, Social Services, Chaplain Services, and Dietary Services provided optimal medical management in a team-based approach. Further studies need to be conducted to explore the effect that daily multidisciplinary rounds have on the outcome of severe TBIs.

Statement

The authors whose names are listed within this manuscript certify they have no affiliations with any organization or group with any financial or non-financial interest in this report.